

A RADIOLOGICAL STUDY OF PNEUMOCONIOSIS  
IN PERSONS WORKING IN STONE CRUSHERS  
IN JHANSI DISTRICT

THESIS  
FOR  
**DOCTOR OF MEDICINE**  
( RADIO-DIAGNOSIS )



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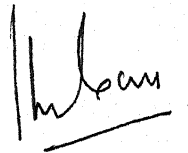
BUNDELKHAND UNIVERSITY  
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C E R T I F I C A T E

This is to certify that the work entitled,  
"A RADIOLOGICAL STUDY OF PNEUMOCONIOSIS IN PERSONS  
WORKING IN STONE CRUSHERS IN JHANSI DISTRICT", has  
been carried out by DR. MAN MOHAN NATH SHARMA himself  
in this department.

He has put in necessary stay in the department  
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19-9-88

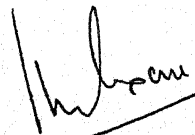


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which is being submitted as a thesis for M.D.  
(Radio-diagnosis) was carried out by DR. MAN MOHAN  
NATH SHARMA under my constant supervision and  
guidance.

The techniques embodied in this work were  
under taken by the candidate himself. The results  
and observations were checked and verified by me  
periodically.



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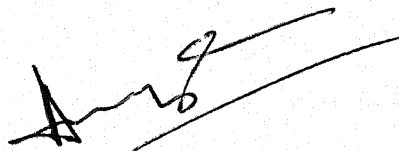
( G U I D E )

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C E R T I F I C A T E

This is to certify that DR. MAN MOHAN NATH SHARMA has worked on " A RADIOLOGICAL STUDY OF PNEUMOCONIOSIS IN PERSONS WORKING IN STONE CRUSHERS IN JHANSI DISTRICT", under my direct supervision and guidance.

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Man Mohan Nath Sharma

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## I N T R O D U C T I O N

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'Pneumoconiosis'-Proust's (1874)

modification of Zenker's term 'pneumonokoniosis' -simply means 'dusty lung'. Thus, any dust-ridden state of the lungs or disease process resulting from it may legitimately be called 'pneumoconiosis'.

Classifying 'pneumoconiosis' for medical purposes rests upon morbid anatomical changes, and thus embraces a variety of lung disorders.

Following this principle the Industrial Injuries Advisory Council (1973) recommended that the terms 'pneumoconiosis' should be taken to mean permanent alteration of lung structure due to the inhalation of mineral dust and the tissue reactions of the lungs to its presence, but should not include bronchitis and emphysema. This recommendation can be usefully expanded as follows: 'Pneumoconiosis' is defined as the non-neoplastic reaction of the lungs to inhaled mineral or organic dust and the resultant alteration in their structure excluding asthma, bronchitis and emphysema. There are three key words in this definition: dust, lungs and reaction.

## DUST

Dusts consist of solid particles of mineral or organic origin dispersed in air and, as such, are distinct from vapours, fumes and smoke although all these categories are commonly embraced by the general term aerosol. Hence, by definition, pneumoconiosis does not include lung disorders such as oedema or pneumonia caused by inhaled aerosols other than dusts.

## LUNG

Strictly anatomically, the term 'lung' does not include the pulmonary (visceral) or parietal pleural membranes which are of different embryological origin from that of the lung parenchyma and, although it is convenient and customary to include the pulmonary pleura in this term, the parietal pleura is clearly distinct. To regard the parietal pleura as 'lung' is contrary to established embryological and anatomical knowledge, and thus, terminologically inaccurate. Hence, disease which arises in, or primarily involves, the parietal pleura-whatever its cause should not be classed or described as 'lung' disease or pneumoconiosis.



## SIZE SELECTIVE DUST SAMPLERS

Human lungs have been aptly called, Size Selective Dust Samplers. Air born dust, when inspired into the lung, undergoes a process of separation based on size and falling rate, like wise breathing patterns and minute ventilation affect penetration and deposition. Over 80% of larger particles ( $6\text{ }\mu$  and over), impact on mucous lining of larger airways to be removed, usually quite rapidly (i.e. within hours) by ciliary escalation however some particles of considerable length, es: Asbestos fibers upto as much as  $100\text{ }\mu$  in length, may penetrate as far as terminal respiratory units. Small dust particles penetrates more deeply but only the fine particles (below  $2\text{ }\mu$ ) penetrates the alveolar spaces. The retention rate is about 40% in  $2 - 1\text{ }\mu$  size and high for those below  $0.2\text{ }\mu$ .

## REACTION

The lungs react to inhaled dust in a variety of ways which are discussed briefly, the reaction may be transient as, for example, in the case of the acute fibrous 'alveolitis' or granuloma formation of farmers' lung, or give rise to permanent reticulin proliferation or collagenous fibrosis.

To avoid misunderstanding at the outset it is important to emphasize that to pathologists, by common consent, 'fibrosis' means excessive production of collagen fibres and not proliferation of reticulin fibres. Dusts which cause fibrosis are termed fibrogenic.

Both inorganic (mineral) and organic dusts may be classified according to the type of reactions they produce. Dust producing permanent fibrosis and disability are classified as malegnant Pneumoconiosis.

The fibrosis which are reversible included in Benign Pneumoconiosis.

The foregoing definition of pneumoconiosis thus includes harmless as well as potentially harmful changes in the lungs and in the following pages it refers both to innocuous dust accumulation and to dust-induced disease confined to the gas exchanging region of the lungs(that is, the acini) but which, in some instances(for example, extrinsic allergic 'alveolitis') may also involve non-respiratory bronchioles.

Improper terminology, sometimes encountered includes the use of 'silicosis' as a general term for all forms of pneumoconiosis, and 'pneumoconiosis' to

refer exclusively to coal pneumoconiosis. 'Mixed dust fibrosis' means pneumoconiosis caused by the combination of free silica with substantial quantities of other dusts such as coal, carbon or iron oxides, and not to different types of pneumoconiosis (for example, silicosis and beryllium disease) in one individual.

During the past 100 years the incidence of silicosis and the pneumoconiosis of coal miners steadily increased in most major industrial countries until the 1950s, since when it has undergone a downward trend, while asbestosis has become increasingly more frequent, and disorders of the extrinsic allergic 'alveolitis' type (hypersensitivity pneumonia) - for example, farmers, lung only recently been properly recognized.

The pneumoconiosis became an important disease when various compensation claims were filed in the court. This led to stimulate various Agencies so that these industrial hazards could be minimised. Various prophylactic measures were devised to reduce the dust particle in the atmosphere. Industries Regular Medical Check ups, Industrial hazard allowance, compensation were.

Introduced workers working in the dusting atmosphere were explained for their hazards.

In short, pneumoconiosis and other dust-induced diseases are an important medical problem from the stand

point of differential diagnosis, and as a cause of respiratory disability and sometimes, premature death in certain occupations.

### ANATOMY

Some familiarity with the basic features of lung anatomy and cytology is necessary for an understanding of the pathogenesis and behaviour of the different types of pneumoconiosis and other occupational lung disease.

### LUNG AIRWAYS

From the trachea downwards each branch of the airways divides progressively into two daughter branches the length and diameter of which are not necessarily uniform. The average diameter of daughter branches is smaller than that of the parent branch but, over the complete number of some 23 generations, the total cross-section and volume of the airways system increase progressively while the individual airways become smaller.

Branchi are characterized by the presence of variable amounts of cartilage in their walls; the continuations, of these airways without cartilage to the alveolar areas of the lung constitute the bronchioles. The last three or four (rarely, up to eight) generations

of bronchioles which carry a variable number of alveoli (alveolus, a little hollow) in their walls are named respiratory bronchioles because they are capable of gas exchange. The terminal bronchiole is the last airway without alveoli before the first respiratory bronchiole. This diagram is not drawn to scale, the airways of the respiratory unit being shown disproportionately large by comparison with the conducting airways, and the distance between generations four and 17 spans the greater length of the lung. The small airways are about 2 mm in diameter or less.

The lining of the airways as far as the terminal bronchiole consists of epithelial cells which are of pseudo-stratified, columnar and ciliated type. Situated irregularly between them are mucus-secreting goblet cells opening to the surface. Goblet cells are plentiful proximally but become progressively fewer in number distally until, in the bronchioles, they are extremely scanty-at least in health.

Mucous glands are found only in bronchi and lie between the epithelium and cartilage. Their total volume is substantially greater than that of the goblet cells and it is likely that they produce the greater part of mucous secretion in health and disease(Reid, 1960). Increased activity is expressed by enlargement. This enlargement is the structural basis of chronic bronchitis, and the comparison of gland thickness to bronchial wall

thickness is a valuable practical index of its presence and degree of severity (Reid, 1960).

Secreted mucus spreads as an uneven layer on the cilia which possess an auto-rhythmic stroke directed proximally and advancing the layer in that direction; this process is often referred to as the 'ciliary escalator's . Although this is an efficient arrangement for removal of inhaled foreign particles it may be impaired or destroyed by some noxious agents, and excess mucus in chronic bronchitis may some-times impose an undue burden upon the cilia.

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REVIEW OF LITERATURE

## REVIEW OF LITERATURE

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### 1. Introduction of dust:

The medical worker in the field of industrial lung disease often encounters mineral and rock names with which he may not be familiar, or he may be unaware of the composition of some well-known substance. A basic knowledge of geology should enable him to decide what the nature and composition of a particular natural mineral or rock is likely to be. This not only saves time and points further enquiry in the right direction but also helps to establish rational thinking about pathogenesis and to avoid mistaken diagnosis.

The earth consists of a superficial crust a few miles thick which rests on a denser mass, the mantle, nearly 2000 miles thick, and a central core which is probably solid but behaves in some respects as if in a molten state. Molten rock material, magma (which contains gases and steam) also exists as pockets within the crust and mantle or is extruded on to its surface as volcanic lava.

Chemically, the 'average' composition of the crust consists of about 27.7 percent silicon, 46.6 per



cent oxygen, 8 per cent aluminium and 16.2 per cent in aggregate of calcium iron, magnesium, potassium and sodium. This gives a total of 98.5 per cent, the remainder consisting of all the other elements.

In our present context it is the crust and its rocks which are of importance. The crust is considered to have an upper and lower zone; the upper zone, which is confined to continents, is composed largely of silica and alumina (SIAL); and the lower zone, which is present beneath both continents and oceans, is predominantly silica and magnesia (SIMA) Rock means 'any mass or aggregate of one or more kinds of minerals or of organic, whether hard and consolidated or soft and incoherent, which owes its origin to the operation of natural causes. Thus granite, basalt, limestone, clay, sand, silt and peat are all equally termed rock' (Geikie, 1908). The ingredients available for rock formation are known as minerals. A mineral is probably best defined as an inorganic homogeneous substance which occurs naturally and has distinct crystal structure, chemical composition and physical properties. Minerals may crystallize in different habits or forms under different physical conditions; for example, prismatic, acicular, asbestiform and platy for single crystalline aggregates (Zoltai and Wyllie, 1979).

Silicon and oxygen are the two most important elements in the crust and form a fundamental  $\text{SiO}_4$  tetrahedral unit consisting of a central silicon ion with oxygen ions attached three-dimensionally at the four 'corners' of a tetrahedron. All forms of 'silica' - that is, silicon dioxide ( $\text{SiO}_2$ )<sub>x</sub> - are composed of these tetrahedra joined by common oxygen atoms so that each crystal consists of a giant molecule with an average stoichiometric formula of  $\text{SiO}_2$ . Being uncombined they are referred to as 'free silica'. The tetrahedra are linked in various ways by Si-O-Si-chains, and the manner in which metallic cations are included in this linkage decides their form and characteristics.

The distinction between 'free' and 'combined' silica is important. Combined silica is  $\text{SiO}_2$  in combination with various cations as silicates. Free silica is the most widespread substance in nature with a fibrogenic potential for the lungs, but examples of combined silica which are fibrogenic (mainly and most importantly the asbestos group of minerals) are of more restricted distribution. It should be noted that many reported chemical analyses of rocks make no distinction between 'combined' and 'free' silica and only the total  $\text{SiO}_2$  content may be shown. Under these circumstances the quantity of 'free' silica remains unknown.

Free silica (silicon dioxide) occurs in three forms: polymorphic crystalline, cryptocrystalline (that is, minute crystals) and amorphous (that is, non-crystalline).

- (1). Quariz which is stable up to  $867^{\circ}\text{C}$  but is capable of metastable existence at higher temperatures;
- (2). Tridymite which is stable from  $867^{\circ}\text{C}$  to  $1470^{\circ}\text{C}$  and capable of metastable existence both above  $1470^{\circ}\text{C}$  and below  $867^{\circ}\text{C}$ ;
- (3). Cristobalite which is stable from  $1470^{\circ}\text{C}$  up to its melting point of  $1723^{\circ}\text{C}$  but is capable of metastable existence at any temperature below  $1470^{\circ}\text{C}$ . Opaline-silica is a variety of cristobalite (Sosman, 1965).

## 2 - Pathogenesis of minerals cause pneumoconiosis:

Precisely why particles of one material provoke collagenous fibrosis or other disease processes-such as sarcoid-type granulomas-whereas those of another do not is poorly understood, but one thing is clear: the form that the lesions of each type of pneumoconiosis takes is primarily determined by particular attributes of the inhaled particles and by cellular and humoral responses to them ; and possibly, concomitant infection in the lung may sometimes play a part. Recent advances in cytology, enzyme chemistry, histochemistry and immunology have identified some important features of the sequence

of pathogenic events following exposure to certain mineral dusts both in man and under experimental conditions.

Many theories to explain the fibrogenesis of different types of pneumoconiosis have been elaborated but only those relevant to current knowledge are discussed.

#### SILECOSIS

##### Theory of piezoelectric effect:

This suggested that minute electrical currents caused by mechanical stresses on quartz crystals may damage tissue cells (Evans and Zeit, 1949), but precisely how was not clear. Against this it was shown that substances other than quartz which possess piezoelectric activity were nonfibrogenic, whereas tridymite, which lacks this activity, is strongly fibrogenic. However, recent work has resuscitated and modified the theory. It is postulated that emission of electrons from the edges of the tetrahedral crystals of silicon dioxide (Chvapil, 1974) or electric charge - transfer between the crystals and cell members (Robock, 1968) initiate the cell damage which provokes fibrogenesis.

##### The solubility theory:

This theory-originally elaborated by Kettle

(1926) and widely favoured until recently - postulated that crystalline silica passes slowly into solution in tissue fluids producing silicic acid which causes fibrosis. However, there are many reasons why this theory is untenable. There is a significant difference in the fibrogenic potential of different types of free silica, the severity and speed of fibrosis being greatest due to tridymite then, in descending order, to cristobalite, quartz and least of all, to vitreous silica although the solubility of all is similar (King et al., 1953; Stober, 1968). Quartz etched with hydrofluoric acid is much less soluble than unetched quartz yet is more fibrogenic (Engelbrecht et al., 1958).

Further more, of the submicron polymorphic forms of silicon dioxide which have similar solubilities to quartz coesite causes little fibrosis and stishovite behaves as an inert dust (Brieger and Gross, 1967; Streacker, 1965). Finally, silicic acid, when allowed to permeate from diffusion chambers into the peritoneal cavities of animals while quartz particles are excluded, does not cause fibrosis (Curran and Rowsell, 1958; Allison Clark and Davis, 1977).

Holt (1957) modified this theory- The so-called extended solubility theory by postulating that silicic acid is absorbed on to the protein of collagen precursors

causing them to polymerize into collagen. But there has been no satisfactory evidence to support this either.

It appear, then that the ability of free silica to cause fibrosis depends fundamentally on two conditions; (1). Particular types of crystal or particle structure and their surface properties; and (2). intimate contact with cells.

The process of fibrogenesis and the control of collagen synthesis are of general medical importance and, for some years past, the effect of free silica on cells in vitro and in animals has been used as a model to investigate this wider problem as well as specifically the genesis of silica - induced disease. In addition, there is now much evidence which points to involvement of immunological processes at some stage in the silicotic process. Hence, there are two, possibly interrelated, concepts of pathogenesis-cellular and immunological - which may also be applicable to other types of pulmonary disease believed to be caused by inhaled aerosols.

Theory based on cytotoxicity and collaboration of macrophages and fibroblasts :

Both non-cytotoxic particles -such as titanium dioxide (rutile), diamond and amorphous carbon-and cytotoxic particles of quartz when added to macrophages in a culture medium containing serum are quickly ingested



by the cells and enveloped in their phagosomes. The resulting digestive vacuoles (secondary lysosomes) which contain non-cytotoxic particles remain intact and the cells undamaged, whereas those which contain quartz soon rupture or become permeable and release their contents into the cytoplasm. There upon, the macrophages become round and immobile, and disintegrate discharging all their contents - particles, enzymes and other constituents which are then re-ingested by other viable macrophages and the process repeated (Allison, Harington and Birbeck, 1966; Nadler and Goldfischer, 1970). A similar train of events has been demonstrated in experimental animal by a double-dusting inhalation technique using both inert and toxic quartz dusts (Heppleston, 1963) though the effects on macrophages are less dramatic since the concentration of mineral particles is generally less than in 'in vitro' experiments (Bruch and Otto, 1967). It seems likely that similar events also occur in man.

Macrophages, therefore, are affected in dissimilar ways by different materials. Inert particles, after phagocytosis do not appear to interfere with the normal life-span of the cells; whereas particles of crystalline silica continue to destroy macrophages until the particles are incarcerated by proliferating collagenous

tissue which presumably prevents a state of perpetual cell destruction. For this reason macrophages probably play a minor role in eliminating quartz and other forms of fibrogenic crystalline silica particles from the lungs.

When macrophages are present by themselves in a millipore diffusion chamber (from which cells or particles larger than 1  $\mu$ m diameter cannot escape) placed in the peritoneal cavity of an animal no fibrosis of the peritoneum occurs, but when both macrophages and quartz particles ( $> 1 \mu$ m diameter) are present together in the chamber significant synthesis of collagen in the visceral and parietal peritoneum results. The degree of fibrogenesis is more pronounced with lower concentrations of quartz which are not rapidly cytotoxic than with higher concentrations which kill most of the cells. These observations imply that macrophages which have ingested an insufficient amount of quartz to kill them secrete a factor which stimulates fibroblasts to synthesize collagen (Allison, Clark and Davies, 1977).

Quartz particles affect macrophages in two distinctly different ways. When the particles are added to the cells in a serum free medium the majority are damaged within an hour due to interaction with the plasma membrane, and lysosomal and cytoplasmic enzymes (such as lactate dehydrogenase) are released. When, however, the



encounter occurs in a medium containing serum the cytotoxic process is delayed because serum proteins coat the particles so that they do not harm the plasma membrane and only when this coating is digested away in secondary lysosomes is the surface of the particles exposed to interact with the lysosomal membrane (Allison, Clark and Davies, 1977). In explanation of this interaction it has been suggested that the numerous strong hydroxyl(silanol) groups of silicic acid on the surface of quartz crystals act as hydrogen donors in hydrogen bonding reactions with the membranes of the secondary lysosomes causing irreversible injury (Allison, 1971); and the importance of the absorption properties of silanol groups in the fibrogenic activity of quartz dusts - which apparently differs in quartz from different geological sites - has, in fact, been demonstrated by physical methods - such as chemoluminescence intensity and infrared spectroscopy (Kriegseis et al., 1977). This harmful effect and that of cell membrane damage is blocked by coating the particles with aluminium or polyvinyl pyridine-N-oxide (PVPNO) (Nash, Allison and Harington, 1966), or by treating macrophages with PVPNO before or shortly after exposure (Allison, Harington and Birbeck, 1966). The polymer enters the secondary lysosomes with the particles and reacts with them when their protein coating has been digested away.

Theory based on immunological reactions:

There are three possible ways in which free silica particles might cause immunological reactions.

(1). By acting as an antigen:

Unlike organic dusts which may provoke an Arthus reaction mineral dusts - including free silica - do not act in this way. Theoretically, they may function as a hapten to produce a type II allergic response, but no satisfactory evidence to support this has been produced. Although some workers, notably Kashimura (1959), have apparently demonstrated antibodies against quartz in experimental animals this observation has not been confirmed (Vioson et al, 1964), and the result of the earlier experiments may have been due to bacterial contamination of quartz particles.

(2). By producing an auto-antigen:

They may modify the structure of some body protein and thereby produce antigen. Proteins absorbed on to the surface of quartz are denatured (Scheel et al, 1954) and by virtue of this, may conceivably acquire antigenic potential. Gamma globulins are most likely to be involved and when denatured in various ways they have been shown to produce antibodies in experimental animals (Milgrom and Witebsky, 1960; Mc Clusky, Miller and Benacekraf, 1962). However, Jones and Heppleston (1961) failed to demonstrate antibodies to whole serum

or  $\gamma$ -globulin by immunofluorescence in experimental silicotic nodules, and there is no convincing evidence of such auto-antigenic activity in man.

Recent work has offered evidence that lung connective tissue antibodies stimulate quartz-exposed macrophages to release a factor which stimulates synthesis of collagen a substance possessing antigenic potential by fibroblasts resulting in the production of more antigen (Lewis and Burrell, 1976). This is of particular interest in view of that fact that the existence of human autoantibodies against denatured collagen now seems to be well authenticated in some disorders associated with collagen breakdown, and it has been suggested that anticollagen antibody might stimulate fibroblasts to synthesize and secrete more collagen (Holborow et al, 1977). If the presence of these events is substantiated they would imply 'collaboration' between the immediate cytotoxic effect of free silica and a secondary and later contributory antigen-antibody reaction.

(3). By acting as an adjuvant:

They may, like Freund's adjuvant, facilitate an allergic reaction which would not otherwise occur (Pernis and Paronetto, 1962). In experimental animals the induction of hypersensitivity causes larger and more clearly demarcated silicotic lesions (due to powdered

quartz) than in control animals (Powell and Gough, 1959).

Although experimental work (Thiart and Engelbrecht , 1967) has indicated that the ground substance of silicotic nodules in rats consists, not of  $\gamma$  - globulins but of  $\beta$ -globulins derived from macrophages killed by quartz, human silicotic nodules apparently contain Ig and 1 g M which are found in the hyaline and among collagen fibres and may be bound to them (Pernis, 1968). Plasma fibres and may be cells around the periphery of actively developing lesions are believed to be the source of the immune globulins. These features vary widely in lesions from different subject and in different lesions in the same subjects: and in some they cannot be identified at all. They appear to be most prominent in actively evolving lesions and absent from old inactive lesions. The nodules contain a variable amount of hyaline in which there is no more than about 40 per cent collagen (Vigliani and Pernis, 1963).

It is possible, therefore, that immunoglobulins may play a role in the evolution of the later stages of silicosis, especially in the formation of conglomerate masses. In this respect it is of interest that amyloid lesions, in which auto-immune phenomena are thought to play a part, also contain variable amount of IgG



(Schultz, et al., 1966) and complement and there are similarities in the hyaline of both types of lesion (Pernis, 1968). However, it may be that the presence of globulins is due simply to permeation of serum proteins which remain sequestered in the lesions (Heppleston, 1969). In view of the lack of agreement in reported observations a possible adjuvant effect of quartz or other types of free silica cannot be said to have been proved.

Inhalation of crystalline silica by mice is reported to result in decreased T-lymphocyte activity in mediastinal lymph nodes but in increased activity in the spleen; and in reduction of B-lymphocyte activity in both of these sites (Miller and Zarkower, 1974a). However, administration by the intravenous route does not apparently demonstrate unequivocal evidence of a direct effect on these cells although macrophage function is, in general, depressed (Levy and Wheelock, 1975). Alveolar macrophages of rats which have inhaled quartz show an increase of surface receptor sites for 1 g G and the C 3 component of complement (Miller and Kagan, 1977). Hence, the possibility that inhalation of free silica might alter the development and expression of acquired immunity is obviously important and continues to be investigated.

### TO SUMMARIZE

The silicotic process appears to be initiated by the cytotoxic effect of free silica on alveolar macrophages and the release of fibrogenic factor(s) from these cells. Antigen-antibody reactions have not until recently, been regarded as playing a pathogenic role in man but there is increasing reason to believe that such reactions may develop at a later stage in the evolution of the disease in some cases. These reactions may be the result of an adjuvant effect or, as seems more likely, of the development of anticollagen antibodies against denatured collagen stimulating increased collagen production.

### (3) Prevalence, Incidence and Progression:

(1) In the late 1940s the prevalence of coal pneumoconiosis in the UK was high due to a lack of adequate dust control measures over preceding years and a greatly increased production drive during the Second World War. However, a great reduction in the levels of airborne dust in the mines was achieved between those years and the early 1960s and, although the problems of dust control were exacerbated by increased mechanization at the coal mines of since 1965, much progress has been made in recent years so that conditions now meet current dust standards in all working areas (National Coal Board, 1977, 1980).

(2) The British National Coal Board (NCB) introduced a five years periodic X-ray schedule for all coal workers in all collieries in 1959 with two purposes in mind: to provide each examinee with the safeguard of regular chest radiographs; and to assess the effectiveness of dust suppression methods. By 1969 it was evident that a higher correlation exists between radiographic changes of pneumoconiosis and dust exposure when the mass of 'respirable' coal mine dust (size range 1 to 5  $\mu\text{m}$ ) is used as an index instead of the number of particles (in the same size range)/ $\text{mm}^3$  of air previously employed (Jacobsen et al, 1971). Hence, gravimetric sampling has been employed in British coal mines since 1970. PMF = Progressive Massive Fibrosis.

(3). In Britain the prevalence of all categories of coal pneumoconiosis in working miners has fallen from 13.4 per cent in 1959-60 to 5.2 per cent in 1978, due mainly to a more than 50 per cent reduction in 'simple' pneumoconiosis; and the prevalence of PMF in 1978 was 0.4 per cent. This trend, which is the result of increasingly effective dust control is also reflected in the numbers of cases first diagnosed by the pneumoconiosis Medical Panels in the UK with the important proviso that these include ex-miners. The slight increase in 1975 was due largely, if not entirely, to the rallying of ex-miners by anew NCB

compensation scheme. Overall prevalence is higher in older (that is, over 44 years of age) than in younger age groups, but has fallen in both over this period and, in South Wales it has always been substantially higher than elsewhere, it has, also shown an encouraging decline (National Coal Board, 1977, 1980).

(4). A similar trend appears to have occurred in the USA where the recent prevalence of all categories is estimated to be 10.1 per cent and of BME, 0.4 per cent (Morgan and Lapp, 1976); in Australia and West Germany, and probably in France and Belgium. However, it is not possible to make valid comparisons of prevalence between different countries because of differences in composition of coal-mine dust, working conditions and, of course, the technique and standard of radiographic surveys and other criteria. There is some evidence that the rank of coal may influence pathogenesis and, therefore, prevalence but the possible importance of non-coal minerals - notably quartz - in mine dust remains controversial (Walton et al, 1977).

(5). The incidence or prevalence rate of 'simple' pneumoconiosis that is, the number of men who develop pneumoconiosis per 1000 workers per year - is related chiefly to the mass of 'respirable' dust over the period of exposure (Walton et al, 1977) which, in turn, correlates



with the amount of coal and other mineral dust in the lungs (Rossiter, 1972a). Smoking habits do not, apparently, modify the attack rate of 'simple' pneumoconiosis (Jacobsen, Burns and Attfield, 1977). The attack rate of PMF is substantially greater in men with category 2 or 3 radiographs than in those with category 1 in whom it is very low.

(6). Progression of pneumoconiosis appears to be related to the category of the radiograph when a man is first seen (Jacobsen, et al., 1971). Recent analysis of the mean dust exposure of individual miners in 20 British collieries has confirmed the correlation between level of exposure and radiographic progression over a ten-year period. Calculations based on analysis of the mean dust concentrations over all coal faces in these collieries showed that, if the concentrations do not exceed  $4.3 \text{ mg/m}^3$  on average over a 35 year period, the probability of category 2 or higher developing should not exceed 2.4 per cent (Jacobsen, 1973 and 1975). This prediction formed the basis of the permitted dust levels introduced in the British Coal mines in 1975. A similar conclusion was reached in Germany by Reisner (1971). Recently this general correlation between exposure to coal-mine dust and 'simple' pneumoconiosis has been confirmed unequivocally though the long-term risks appear to have been underestimated by 1 to 2 per cent age probability units in the earlier computation (Jacobsen, 1979).

(7). A recent detailed 20 year follow-up study of miners and ex-miners in South Wales (Rhondda Fach) has shown that those with 'simple' pneumoconiosis (categories 1, 2 and 3) and category A opacities survive as well as those with no evidence of pneumoconiosis (category - 0) (Cochrane et al, 1979). This confirms the findings of earlier surveys in Britain (Higgins et al, 1968a; Cochrane, 1973, Cochrane and Moore, 1978) and in the USA where the life expectancy of miners as a whole is the same as that of the general population (Ortmeyer et al, 1974). Interestingly among Pennsylvanian miners with category B pneumoconiosis mortality is higher in those who worked in anthracite mines than in those in bituminous mines (Ortmeyer, Baier and Crawford, 1973). In 88 miners and ex-miners with category B or C pneumoconiosis who died in the quinquennium 1966-1970 the average age at death from all causes was 70 years, the range being 53 to 85 years (Parkes, 1972); and Rooke et al, (1979) found that the mean age of death among Lancashire miners with PMF was 72 years. In a group of 346 South Wales miners and Ex-miners with category B or C pneumoconiosis death was attributable to the pneumoconiosis in about one-third of cases (Sadler, 1974).

It can be concluded that 'simple' pneumoconiosis does not curtail life expectancy, and advanced PMF

affects mortality in only a minority of cases.

#### REFERENCES

##### 1. Classification of pneumoconiosis Radiographs:

It is important that the technique of interpreting as well as taking films should, as far as is practicable, be standardized-especially in the case of epidemiological surveys in order to reduce to a minimum discrepancies between different observers. Hence various systems of classification have been proposed to standardize description of opacities but that proposed by the International Labour Office in 1959 and its subsequent modifications in 1970 and 1971 with sets of standard reference films has been generally adopted (Jacobsen and Gilson, 1972; Liddell, 1972). In 1980 a further modification with a new set of standard films has been introduced. The classification is designed to provide a means for recording systematically the radiographic abnormalities in the chest provoked by the inhalation of dusts seen in standard (PA) radiographs (International Labour Office, 1980). In the main it seeks to do two things: to categorize the size or form of opacities and to indicate their profusion or extent in the lung fields.

2. The ancient occupation of quarrying and fashioning sandstone grindstones in ill-ventilated pits and caves in northern Nigeria has resulted in a 39 per cent prevalence rate of silicosis (Warrell et al, 1975), and the disease is also found in various stages among women in the Transkei District of South Africa who have used similar stones for many years to grind maize and corn (Palmer and Daynes, 1967). A recent survey of stone cutters in North India revealed a 35.2 per cent prevalence (Gupta et al, 1972).

3. A survey in the diatomite processing industry in 1953 - 1954 showed that 25 per cent of 251 workers with more than 5 years' dust exposure, and nearly 50 per cent of 101 workers exposed to high concentrations of calcined dust had radiographic evidence of pneumoconiosis which, in the main indicated nodular and confluent lesions. The majority of these employees has been mill hands handling calcined material. There were no cases among the quarry workers (Cooper and Cralley, 1958).

4. Another radiographic survey of 869 diatomite workers revealed that of those who had been mill hands for more than five years 17 per cent had 'linear - nodular' (Simple) pneumoconiosis and 23.2 per cent larger confluent opacities (Oechsli, Jacobson and Brodeur, 1961).

5. Rigorous dust controls in quarrying and processing

plants, however, have resulted in the virtual elimination of new cases. For example, by 1974, 14 (3.3 per cent) of a work force of 428 men employed since 1853 in an American plant where these measures had been introduced in the mid- 1950s has radiographic evidence of pneumoconiosis and this did not exceed ILO category 1/1. Of 129 employees exposed before 1953 only two mill workers (2.6 per cent) had category A opacities thought to indicate pneumoconiosis (Cooper and Jacobson, 1977).

6. Comprehensive studies have been done by Motley, Smart and Valeo (1956) and Motley (1960). As in the case of other types of pneumoconiosis, abnormal values correlate poorly with radiographic appearances and good pulmonary function may be associated with fairly extensive radiographic changes, but large confluent lesions are usually associated with abnormal function.

7. The earliest abnormality consists of linear or round (nodular) opacities, or both (Linear - nodular) in the upper and mid-zones of the lung fields and extending to their periphery. These appearances are sometimes fine and 'lace-like'. It is unusual for the discrete round opacities to exceed about 2 mm in diameter and they have low contrast with the surrounding tissues, rarely possessing the radiodensity of those due to nodular silicosis (Oechli, Jacobson, and Brodeur, 1961).



8. Surveys of workers in the Hi-sil, Silene and Aerosil processes, in which men were observed over periods of eight to 12 years, revealed no evidence of pneumoconiosis or harmful effects (Plunkett and De Witt, 1962; Volk, 1960).

9. A report that 'amorphous silica dust' of 0.05 to 0.75  $\mu\text{m}$  particle size originating from an electric -arc furnace in a metallurgical process in which quartz was vaporized at temperatures in excess of 2350°C caused 'nodular' fibrotic pneumoconiosis can hardly be accepted in view of the fact that the dust from this plant - which was shown to provoke pulmonary fibrosis of similar severity to that of quartz in animals - was, as the authors themselves state, identified by the American National Institute of Occupational Safety and Health (NIOSH) as cristobalite with a layer of amorphous silica (Johnson, Lewis and Groth, 1973; Vitums, et al, 1977).

(4) Association of other Disease along with Pneumoconiosis  
Clinical features:

Symptoms:

It is important to emphasize that there may be no symptoms even though the radiographic appearances may be surprisingly advanced.

Cough may develop as the disease advances and is

of variable, mainly in the mornings but sometimes intermittently throughout the day and night. In the later stages there may be prolonged and distressing paroxysms due, possibly, to irritation of nerve receptors in the trachea and bronchi by silicotic lymph node masses.

Often there is no sputum or only a small quantity of mucoid appearance raised from time to time during the day. However, in advanced disease recurrent bronchial infections tend to occur and produce quite a large volume of purulent sputum. There is no haemoptysis in the absence of other complicating disease.

Unless there is accompanying chronic obstructive bronchitis of allergic asthma there is no wheeze, although some patients who have narrowing, distortion and fixity of the trachea and main bronchi caused by contiguous silicotic nodes may complain of stridor, especially during effort when there is increased velocity of air flow. This is an uncommon symptom.

Breathlessness occurs as the disease advances, first during pronounced effort and later with lesser degrees of effort; it is rarely complained of at rest unless other lung disease is present. The presence and severity of dyspnoea and impairment of lung function correlates poorly with radiographic appearances.

Chest pain is not a feature of silicosis.

General health is unimpaired unless tuberculosis or congestive heart failure supervenes. Haemoptysis and loss of weight may signal the presence of tuberculosis.

Physical signs:

The general physical condition is good but deteriorates with the onset of congestive heart failure and in the presence of tuberculosis. Central cyanosis is absent unless there is complicating heart or lung disease, and dyspnoea at rest suggests disease other than silicosis.

Finger clubbing is not caused by silicosis and when observed is either of congenital type or evidence of other pathology.

The chest contour is usually normal but in advanced disease there may be localized flattening of one upper zone possibly with some degree of dorsal scoliosis. Expansion remains good and equal until a late stage of the disease when it may be somewhat diminished often on one side (where underlying fibrosis is greater) more than the other.

The trachea is some times displaced to one side either by silicotic hilar masses or a large distorted conglomerate mass in an upper lobe. Occasionally, hard, non-tender, silicotic lymph nodes are palpable in the neck and supraclavicular fossae.



Percussion note is unaffected unless there are areas of unusually dense pleural fibrosis-chiefly in the upper zones.

Breath sounds are normal or reduced by pleural thickening and inspiratory and expiratory stridor (of greater or lesser intensity) may be heard over the trachea and at the open mouth when there is excessive distortion of trachea or main bronchi; when this sign is present it is persistent.

Adventitious sounds are not heard in disease uncomplicated by chronic obstructive bronchitis or tuberculosis.

In the advanced stage of silicosis the signs of pulmonary heart disease may eventually develop with or those of congestive heart failure.

### Investigations

#### Lung Function:

In the early radiographic stages (ILO category 2 to 3) impairment of any parameter of lung function is generally absent but in some cases slight reduction in VC and of arterial oxygen tension (on effort) may be observed. With more advanced disease, impairment is commonly present but often of a much less degree than the radiographic category

might suggest. There is a decrease of TLC, VC, RV, FRC and compliance without evidence of airways obstruction and, in some cases, a slight reduction in gas transfer although this is often remarkably little affected even in the presence of advanced disease. Oxygen desaturation is not present at rest or on moderate effort (300 kg.m/min) in the non-conglomerate stage of disease (Becklake, de Preez and Lutz, 1958), but may be observed on greater effort in some cases. As the disease progresses to massive conglomeration in equality of gas distribution and of ventilation-perfusion ratio occurs resulting in some impairment of TI in addition to the volume changes mentioned. However, in a study of non-smoking men with non-conglomerate ('simple') silicosis TI was not reduced and even in these with Category B and C conglomeration KCO was normal in most cases (Teculesscu and Stanescu, 1970). Ventilation - perfusion imbalance and arterial oxygen desaturation on effort are determined by the extent of arteritis as well as by silicotic fibrosis.

The best overall guide to the degree of respiratory disability in conglomerate disease is the ventilatory capacity.

There is nothing characteristic in the patterns of impaired function in silicosis.

Radiographic appearances:

The earliest radiographic evidence of nodular silicosis of small discrete opacities of moderate radiodensity which appear in the upper halves of the lung fields and vary from 1 to 3 m m diameter (ILO category 'p' and 'q'). It has been claimed, however that linear opacities accompanying the normal vascular markings are the earliest evidence of silicosis but this is not generally accepted (Ashford and Enterline, 1966), and appearances (of which it is difficult to be convinced do not correlate with morbid anatomical evidence of silicosis.

There appears to be a clear relationship between total dust exposure and radiographic evidence silicosis (Beadle, 1971).

As the disease advances, discrete opacities increase in number and size and occupy the lower as well as the other zones of the lung fields. In general they are roughly symmetrical in the two fields but are sometimes of disparate size and distribution. Small conglomerations may then appear-usually, but not always, in the upper zones and subsequently develop into large, irregular and some times massive opacities which may occupy the greater part of both lung fields. Bullae may be seen in the vicinity of conglomerations and in some cases there may be

significant bowing and distortion of the trachea. Cavity formation in the absence of complicating tuberculosis is uncommon. Rarely, unilateral conglomeration is present in the absence of other evidence of silicosis. The curious tendency for the silicotic masses in Lipari pumice workers to occupy the lower lung fields. Occasionally the discrete opacities are small and very dense and may closely resemble the appearances of the calcified nodules seen in some case of 'rheumatoid' coal pneumoconiosis and in microlithiasis.

An important feature of some cases is evidence of lymph node calcification which is characterized by thin, very dense ring shadows around the nodes - so - called 'egg-shell' calcification. Nodes most commonly involved are those of the hilar and mediastinal groups but other intrathoracic nodes (for example, the internal mammary chain) and extrathoracic nodes (notably the supraclavicular, cervical and axillary groups and occasionally the intra - abdominal and inguinal groups) may also be affected (polacheck and Pijanowski, 1960). Curiously, there appears to be no correlation between the intensity of the calcification and the amount of silicosis or the presence of pulmonary tuberculosis (Chiesura, Terribile and Bardellini, 1968). Calcification

may be prominent when there is little or no obvious lung involvement, or absent in the presence of advanced silicosis. Although not a pathognomonic sign - as it is seen occasionally in sarcoidosis, tuberculosis, and histoplasmosis - predominantly peripheral calcification of hilar nodes is highly suggestive of silicosis or of exposure to some form of free silica.

Evidence of diffuse bilateral pleural fibrosis is present in many cases and in advanced disease may be extensive and partly calcified; occasionally calcified thickening occurs with little obvious lung disease.

The radiographic appearances of the rare cases of silicosis which develop exceptionally quickly bear little resemblance to those of typical silicosis. Opacities are numerous, widespread, small and ill-defined, and spontaneous pneumothoraces are apt to occur.

Though the different patterns of behaviour of silicosis appear to be determined mainly by the amount of siliceous dust inhaled, idiosyncratic reaction seems to be involved. In general sudden changes are most to be due to complicating tuberculosis but occasionally they are associated with rheumatoid disease. The typical appearances of Caplan-type necrobiotic nodules are very uncommon, but have been



reported (Chiesura, Bruguone and Mezzanotte, 1961; Gambini, Agnoletto and Magistretti, 1964; Lamvik, 1963). Occasionally large, ill defined opacities develop rapidly, exhibit vacilliant behaviour over subsequent years, and are sometimes associated with recurrent, transient pleural effusions in individuals with active rheumatoid arthritis or with high titres of circulating RF without overt arthropathy.

It is rarely necessary to use radiographic techniques other than PA , AP and lateral views for diagnosis, though tomography may be helpful in demonstrating silico-tuberculous cavities. Bronchography may reveal bronchial distortion, filling defects and localized bronchiectasis in cases with conglomerate masses.

Bronchial arteriography demonstrates the presence of bronchial artery enlargement and bronchopulmonary shunts in areas of conglomerate masses but not in discrete nodular disease (Tada et al., 1974).

#### Other investigations:

Biopsy of lung tissue should not be required for diagnosis, although examination of a scalene lymph node may resolve occasional problematical cases by demonstrating silicotic nodules.

It is advisable to obtain sample of sputum for culture of *M. tuberculosis* periodically, for life, as

tuberculosis may develop at any time.

Tests for circulating rheumatoid and antinuclear antibodies positive, suggest the possibility of an underlying immunological cause for suddenly advancing tuberculosis negative silicosis.

Electrocardiography may be required in advanced cases to establish or refute the presence of cor pulmonale.

Investigations for sarcoidosis may be indicated on rare occasions to distinguish the two diseases.

The erythrocyte sedimentation rate is not raised in the absence of complicating tuberculosis or other disease.

#### Diagnosis:

When a satisfactory occupational history is combined with good quality radiographs, nodular silicosis should rarely be mistaken for the diseases. The chief cause of misdiagnosis is failure to recognize the 'silica' hazard of a past occupation, and the fact that the prevalence of silicosis is now low increases the possibility of this error.

An isolated conglomerate lesion (so-called 'silicoma') may be confused with bronchial carcinoma,

especially in a first radiograph. Tomography may help in the distinction by revealing multiple small densities within silicotic mass.

When silicotic lung lesions and hilar and pulmonary lymph nodes are calcified it is necessary to exclude.

Malignancy:

Firstly, tobacco, smoke is established as the over-whilmingly dominant respiratory carcinogen in man but certain minerals and chemicals are known to cause lung cancer both in smoker and non-smokers though the risk is substantially greater in smokers - in short, a cocarcinogenic effect. This excess risk in smokers applies to all known industrial carcinogens. However, the available evidence indicates that industrial carcinogens are responsible for only a small proportion of lung cancers, for the occupational component of all cancers is estimated to be approximately 1 to 5 per cent (Higginson and Muir, 1976; Doll, 1977; Wynder and Gori, 1977; Higginson, 1980) - possibly as much as 15 per cent in the USA (Cole, 1977). Nonetheless, this group, though a minority is important. A recent report by Bridbord et al, (1978), which claims that some 30 per cent of all cancers which develop in the next few decades will probably be wholly or partly due to industrial carcinogens, has been strongly criticized as being based on evidence which is inadequate in a variety



of important respects (Leading article, 1978; Abelson, 1979; Morgan, 1979). Higginson and Muir (1979) maintain that on present knowledge, 'most cancers of presumed environmental aetiology can not readily be ascribed to industrial exposures, either point source or general!

Secondly, epidemiological studies of cancer incidence in industry must have cohorts and mortality data (including hospital and post-mortem records) which are validly matched in all relevant respects and cover a sufficient period of time. This is important in view of the relatively low attack rate of carcinoma of the lung and the long interval between cessation of exposure and development of the tumour in most cases (Kotin, 1968).

Surveys in England and Wales have shown that, in spite of the fact that certain occupations are associated with a greater or lesser cancer risk than the general population average, almost 90 per cent such variation disappears when comparison is made between individuals of similar habits and social class (Registrar general 1978; Fox and Adelstein, 1978; Higginson, 1980a). The 88 to 90 per cent of cancers which are of non-occupational origin are related to so-called 'lifestyle' factors - 'lifestyle' being understood as 'the total cultural, behavioural and dietary environment'. It is unlikely that carcinogenesis can be conceived of in

terms of any single mechanism , though one may be overwhelmingly predominant. 'Thus, over 80 per cent of lung cancers in men would not occur in the absence of the cigarette habit. In this sense, cigarette smoking may be considered the practical cause without precluding a modulating role for other factors, including individual susceptibility' (Higginson, 1980b). At present there is some controversy regarding the 'lifestyle' concept of carcinogenesis- the opposing aspects of which are exemplified by Epstein and Swartz (1981) and Peto (1980)- but it is not appropriate to discuss this here.

Thirdly, although animal experiments may give advanced warning of possible carcinogenic potential in man, extrapolation of tumour induction in animal to man involves many pitfalls (such as doses of the suspected carcinogen administered , species response differences and spontaneously developing tumour) and is rarely valid in the absence of clear epidemiological confirmation in exposed workers (Morgan, 1978). Whether or not in vitro testing of bacterial mutagenesis in the presence of a suspected carcinogen will prove to be more validly applicable to man remains to be determined (Bridges and Fry, 1978). Although rigorous epidemiological methods are essential for detection of a cancer risk of low magnitude - as in respiratory cancers induced by ionizing radiation in some circumstances - 'the alert clinician remains the most important source of leads to occupational cancer'

(Archer, 1977; Cole, 1977).

### Complications

#### Carcinoma of the lung :

Wood and Gloyne (1934) first suggested that individuals with asbestosis might develop carcinoma of the lung and Merewether (1949), in the UK, reported that 14.7 per cent of men with asbestosis died of lung cancer between 1926 and 1947. The relationship was confirmed by Doll (1955) who showed that asbestos textile workers with asbestosis and ten or more years exposure had a ten-fold excess risk of acquiring the disease. By 1955, 39.4 per cent of individual's with asbestosis died of carcinoma of the lung (21.6 per cent of males, 17.8 per cent of females) and at the end of 1963 just over 50 per cent died of an intrathoracic neoplasm (including four recorded 'mesothelioma of pleura') (Buchanan 1965). A similar trend has been observed in the USA and elsewhere. This increase is attributable (at least in the UK to a combination of improved life expectancy of individual with asbestosis whose dust exposure during the 1930a to the 1950a was, in general worse than it has been since and to the steady rise in cigarette consumption since the 1920s. It appears that in individuals with asbestosis in Britain, who smoke, life expectancy is reduce by about ten years compared with non smokers due, mainly , to brochial carcinoma (Berry, 1981).

However, rigorous hygiene measures in a British asbestos textile factory over approximately 30 years following implementation of Asbestos Industry Regulations (1931) in 1932 resulted in there being little increase in mortality from bronchial carcinoma among workers who entered the industry after 1932 (Knox, et al, 1968), although a further eight and a half year follow-up of this group has shown a slight increase in mortality from the tumour (Peto, et al, 1977).

As a rule the tumour arises in the vicinity of the lungs-usually the lower lobes (Hueper, 1966; Jacob and Anspach, 1965; Whitwell, Newhouse and Bennett, 1974). It may be of bronchial (Central) or peripheral origin and its commonest histological appears to be adenocarcinoma which is more frequently peripheral than are the other types (Hourihane and Mc Caughey, 1966; Hueper, 1966; Whitwell Newhouse and Bennett, 1974). Animals exposed to asbestos have shown a greater frequency of adenocarcinoma than squamous carcinoma with Canadian and Rhodesian Chrysotile and an equal frequency of both tumours with other types of fibre (Wagner, et al, 1974). Kannerstein and Churg (1974) however, did not find an over all excess of adenocarcinoma in a group of asbestos workers compared with a control series but, as Whitwell, Newhouse and Bennett (1974) point out, it is difficult to assess the significance of their findings which were based on disparate material derived from bronchial biopsy, secondary deposit

biopsy, surgical specimens and post-mortem tissue.

Not unexpectedly the frequency of lung cancer is increased in the smokers compared with non-smokers (Whitwell, Newhouse and Bennett, 1974) but as most of the information on this topic in asbestos workers has been derived in the main from epidemiological and not post-mortem studies information concerning the presence or absence of asbestosis has been inadequate. The effects of smoking and asbestosis appear to be synergistic and multiplicative (Saracci, 1977).

Although a relationship between asbestosis and carcinoma of the lung is well established in man and supported by observations in animals (Wagner et al, 1974) there is some difficulty in regarding the fibrosis as directly responsible for the tumour because an excess of lung cancer does not occur with other fibrotic pneumoconiosis. But on the other hand, there is an increased risk of bronchial carcinoma in cryptogenic fibrosing 'alveolitis', 'honeycomb lung' and DIPF associated with the 'collagen disease' (Meyer and Liebow, 1965; Godeau et al, 1974; Turner-Warwick, Burrows and Johnson, 1980). The apparent link between impaired T lymphocyte function and clinical asbestosis (especially when advanced) in the absence of any evidence of lung cancer raises the possibility of reduced immune ('Killer cell') surveillance and destruction of potentially



neoplastic cells in asbestosis and a consequent increased propensity to develop malignancy. By contrast, patients with malignant pleural mesothelioma apparently have normal T lymphocyte responses and delayed hypersensitivity (Haslam et al., 1978; Pierce and Turner -Warwick, 1980).

The possibility, therefore, that asbestosis is associated with a reduced ability for the lung to destroy potentially malignant cells requires to be explored by detailed longitudinal studies of individuals with asbestosis and matched control from the point of view of the function of lymphocyte populations in bronchial washings and in the peripheral blood, especially as there is evidence that T lymphocyte function in the lungs of smokers without asbestosis is impaired by comparison with the function of these cells in the peripheral blood (Daniele et al, 1977).

This risk of bronchial carcinoma complicating asbestosis must always be borne in mind especially in individuals who smoke and have had long exposure to asbestosis. In order to identify the growth as early as possible it is advisable to examine workers with more than ten years exposure to asbestos routinely at yearly intervals.

#### Evidence in man:

Malignant mesothelioma of the pleura was first related to exposure to crocidolite in the north -western Cape Province of South Africa and in Western Australia

(Wagner, Sleggs and Marchand, 1960, McNulty, 1968) and this has been confirmed among women who worked filling gas mask filters with pure crocidolite during the Second World War (Jones Pooley and Smith, 1976). (Occupational exposures to pure chrysotile has been very uncommon but, when experienced does not seem to have been associated with mesotheliomas (Elwood and Cochrane, 1964; McDonald, 1973b; Weis, 1977). However, a very low incidence of the tumour has been reported in workers apparently exposed to pure chrysotile at the major mine in Quebec Province (McDonald, and McDonald 1977b), but when amphibole as well as chrysotile, fibres were found in the lungs of some workers from this site (Pooley, 1976) it was discovered that crocidolite had in fact been processed there for gas mask manufacture during the Second World War. However, there is now convincing evidence that amosite may be responsible for some mesotheliomas in the USA (McDonald, 1980; McDonald and McDonald 1980). Pure amosite exposure has been held responsible for the occurrence of mesothelioma in a group of insulation workers in the USA (Selikoff et al., 1972). But the only source of supply of this fibre during the relevant period was the Eastern Transvaal where crocidolite and amosite are intimately associated. The pleural tumour has also been reported in association with exposure to fibrous tremolite present in outcropping mineral deposits used in whitewash and stucco for the walls and roofs of houses in certain districts in Turkey (Yazicioglu et al., 1980). No confirmed

cases of mesothelioma appear to have resulted from pure anthophyllite exposure (Meurman, Kiviluoto and Hakama, 1974).

It is often claimed, as was originally thought that malignant mesothelioma follows trivial exposure to asbestos in the remote past but both epidemiological and pathological evidence has clearly shown that it is, in fact, dose-related - though not so strongly as asbestosis. The tumour rate tends to increase with intensity and duration may have been short in some cases, it has usually been intense (Newhouse 1973; Newhouse and Berry, 1976; Peto, 1979). A dose relation-ship is substantiated by the fact that the content of asbestos fibres in the lungs of mesothelioma cases is significantly higher than in non-mesothelioma cases and amphibole fibres are predominant (Pooley, 1973b; Whitwell Scott, and Grimshaw, 1977). In general however exposure levels tend to be less than those which produce asbestosis.

The paradoxical situation in which there is a clear association between mesothelioma and exposure to crocidolite dust in the Cape Province whereas the occurrence of tumours following exposure to the geologically intimately related crocidolite and amosite of the Transvaal is very rare has been explained by the fact that both the diameter and length of the Transvaal fibres are on average three times greater than the Cape fibres which imposes difference in aerodynamic behaviour Fewer 'respirable' fibres



is Transvaal asbestos are likely to be liberated and they have a higher settling rate and , hence , may be less likely to reach the periphery of the lung. The fibres of Australian crocidolite which is also associated with malignant mesotelioma (Jones, Pooley and Smith, 1976; Miline, 1976) are even smaller in diameter than those of caps crocidolite (Timbrell, 1973).

There is no evidence that mesotheliomas of the pleura originate in parietal pliural plaques; and this is supported by radiographic evidence of bilateral calcified plaques or changes of asbestosis being found in only 12 percent of one large series of mesothelioma cases (Elmes and Simpsom, 1976).

Cigarette smoking is not related to the development of this tumour.

Preliminary studies of cellular immunity in mesothelioma of the pleura have produced some what contradictory results, Haslam et al, (1978) found no evidence of impaired T lymphocyte function either in vitro by PHA stimulation or in vivo by delayed hypersensitivity skin tensting though Wagner (1978) has reported depressed lymphocyte transformation to PHA.

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## MATERIAL AND METHODS

## M A T E R I A L   A N D   M E T H O D S

Diagnosis of Pneumoconiosis depend as the occupational history. Clinical symptomatology, and Diagnostic Procedure e.g. Radiological examination, lung function test, X-ray examination is very important because it tells the extent of involvement. Various type of presentation, whether the change are reversible, which diagnosed after serial radiographs after removal of occupational Pollutants.

When the pneumoconiosis are under consideration it is particularly important that certain principles which determine the appearance of the chest radiograph should be understood if errors of interpretation are to be avoided.

Chest radiography is employed both for clinical and epidemiological purposes: the first to establish diagnosis, prognosis and guidance of treatment; the second to estimate prevalence and behaviour of disease in different industries. Our concern here is mainly with the first. Whichever the purpose, however, the highest possible standard of radiographic technique must always be sought.

### THE STANDARD CHEST RADIOGRAPH

Conventionally, the routine chest film is a postero-anterior (PA) view and is usually 12 x 15 inches. During X-ray exposure the subject's breath must be held in the deepest possible inspiration the mid-expiratory position suggested by the International Labour Office (1959) is not desirable as it is not capable of accurate control and prevents full expansion and, hence, optimal visualization of the lung fields - a matter of importance in the examination of asbestosis subjects. The technique for taking good quality films is summarized in International classification of Radiographs of Pneumoconiosis (ILO, 1970). Too white (under exposed) or too dark (over-exposed) films are to be avoided, and the quality of serial films should, as far as possible, be kept comparable. The kilovoltage range in common use is 60 to 80 kv and with standard speed films it produces high quality pictures providing exposure time is less than about 0.08s. It is claimed that the quality and detail of the standard posteroanterior view is increased by using 110 to 140 kv range and a very fine, stationary lead grid (Jackson, Bohlig and Kiviluoto), but this technique is not in general use.

The subjects should be unclothed above the waist, and corsets and lumber belts removed because, in addition to obscuring the lower lung fields, these limit maximal

inspiratory descent of the diaphragm. Failure to remove clothing may lead to confusing artefacts which are often subtle so that vigilance is required if they are not to be interpreted as evidence of pathology - especially if the obvious clue of pins and buttons is absent and it is taken for granted that the subject was stripped to the waist when the film was taken. This problem is more likely to be encountered in periodic radiographic examinations of workers in industry than in clinical practice.

Consistency of technique is essential at all times in order to detect early radiographic changes and maintain a good comparative standard. This is particularly important in monitoring working industrial populations over a period of years.

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OBSERVATIONS

The costal margins of the lung fields should be followed from the lung spices to the costophrenic angles and then along the diaphragm to the cardiophrenic angles. In this respect one should be familiar with the lower 'Companion shadows' of the lateral chest wall which may be seen for a few rib spaces above the costophrenic angles in a proportion of normal PA films. These are triangular opacities whose lateral aspects are continuous with the rib shadows and their medial aspects usually well defined and vertical, while their lower parts lack definition. They are bilateral although not necessarily symmetrically equal and are caused by the interdigitations of the serratus anterior and external abdominal oblique muscles. Slight rotation of the chest during exposure of the films makes these shadows more prominent on one side than the other, when they may be misinterpreted as pleural lesions if their true nature is not recognized. However, they disappear completely on oblique views.

Stage 2 - Central Region:

The position, size and shape of the trachea, great vessels and heart are noted. The heart shadow, especially on the left, must be 'looked through' for any abnormal opacity which may be superimposed upon it.



Stage 3 - Hilar Region:

The position, size and shape of the hilar shadows - which are caused by the basal pulmonary artery and proximal parts of the pulmonary veins - are examined and the size and distribution of the pulmonary arteries passing to the lung fields noted.

Stage 4 - Lung fields:

These should be examined in two stages. First, the pulmonary arteries ('lung markings') should be followed until they are no longer visible which is usually in the outer third of the lung fields, and their branching and size (whether unduly thick or narrow) noted. These appearances are produced by vessels which run roughly parallel to the plane of the film, but those running in the anterior-posterior plane, and at right angles to that of the film, appear as oval or round opacities. If this is not understood they are sometimes wrongly interpreted as round lesions of silicosis (or some other discrete pneumoconiosis) when there is a relevant occupational history.

Second, the vessels must, as far as possible, be ignored and the lung fields, 'looked into' for opacities indicating lesions in the parenchyma, and their size, shape and distribution assessed.

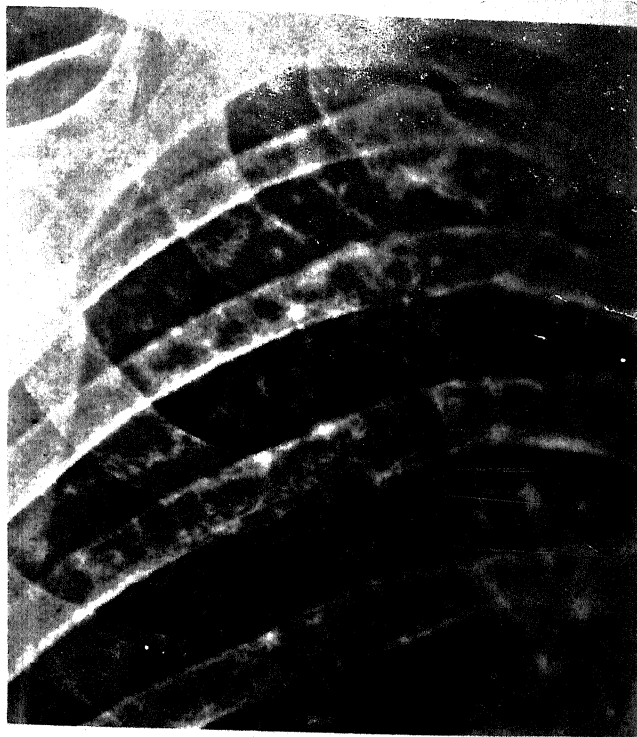
Corresponding regions in both fields must be compared throughout.

Each lung field is arbitrarily, but conveniently, sub-divided into three zones by two horizontal lines, drawn respectively through the anterior ends of the second and fourth ribs. These demarcate upper, middle and lower zones to which the distribution of abnormal shadows can be referred.

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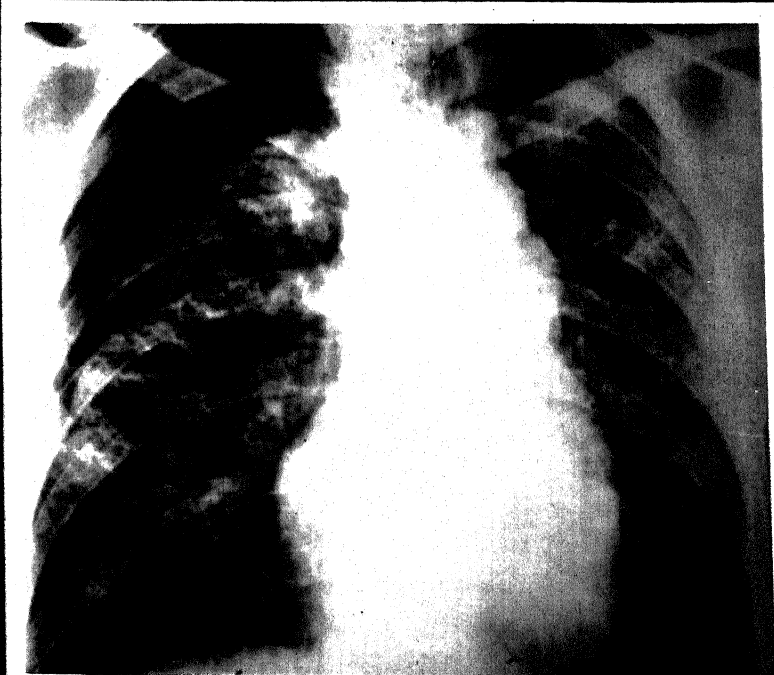
1. Discrete radiographic opacities of typical  
Pneumoconiosis at an early stage (Natural Size).



2. Progression of discrete 'Nodules' of Pneumoconiosis  
from low category to over 10 years.



4. Massive Pneumoconiotic conglomeration with bi-lateral upper zone pleural thickening and displacement of Trachea to the Right. Crusherman in Stonecrusher 15-18 years.



5. Opacities are numerous but small and indefinite with Bilateral Pneumothorax.



6. Irregular opacities presents in both lower zones.

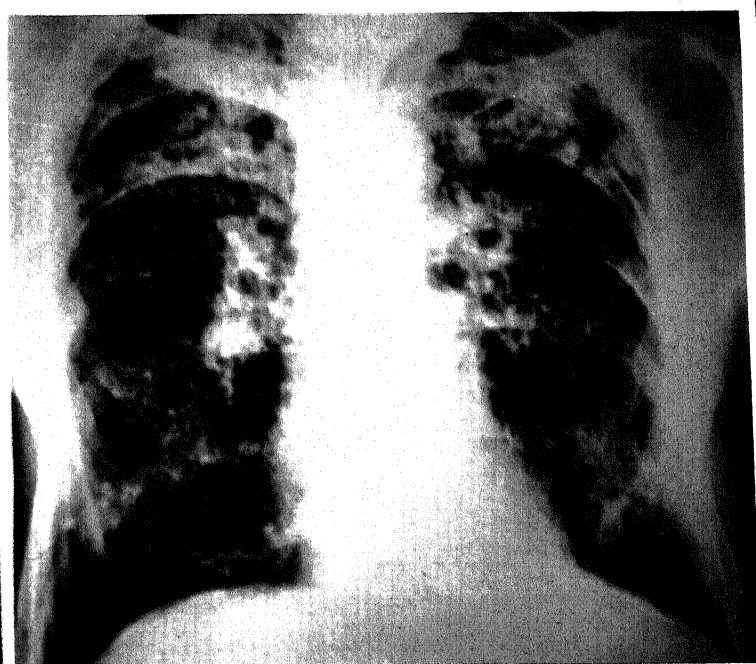


7. Pneumoconiosis with small and large mixed opacities.  
Calcification of some lesions also seen. Family  
History of Rheumatoid Arthritis.





8. Peripheral 'Mixed' type of Pneumoconiosis in man exposed to dust cavities are present in some lesions and there is some Pleural thickening. No evidence of Pulmonary Tuberculoses.



9. Pneumoconiosis in a crusher worker for 18 years. Note dense linear opacities, Calcification on both leaves of the diaphragm.



## DISCUSSION

The modern substitute for white Portland Cement, for construction of building is extensively manufactured & used in many countries of the World. The employee in manufacture of Portland Cement are frequently exposed to dust. Gardner and Others (1939) made a survey of 17 cement workers employing 2,278 men. Dust contain from 1 - 30% of silica. X-Ray chest show nodular silicosis in only 8 out of 2,278 employees, and in 6 of these exposed to silica dust. The incidence of tuberculosis and other chronic affection of lungs was found. It was concluded that Prolonged inhalation of dust does not predispose to tuberculosis. In general, Gardner considered that the problem of dust disease of lungs in cement industry minimal.

Agricola (1556) strongly advocated the ventilation of mines, for he know that dust entering the lungs caused disease associated with dyspnoea corrosive dusts which ulcerated the lungs, producing consumption.

Isbrand Van Die Merbroeck (1609-79) Professor of Medicine in Utrecht, described how several stone cutters died of Asthma and he found at necropsy that to cut their lungs was like cutting a mass of sand and how stone cutters

breathe in small splinters and turn asthmatic and consumptive.

In 1943, calvert Holland described the conditions of work amongst grinders of drystone and dust rose in clouds pervading the atmosphere in which they were confined. He discovered on examination that among 97 men, 30 were suffering from Asthama. He recorded 61 died in 15 years aged 35 to 30 years of age.

The dust, which is thus every moment inhaled, gradually undermines the vigour of the constitutions, and produces permanent disease of the lungs, accompanied by difficulty of breathing, cough and a wasting of the animal frame, often at the early age of twenty-five. Such is the destructive tendency of the occupation, that grinders in other departments frequently refuse to work in the same room, and many sick clubs have an especial rule against the admission of dry grinders generally, as they would draw largely on the funds from frequent and long continued sickness. In 1,000 deaths of persons above 20 years of age, the proportion between 20 and 29 years, in England and Wales, is annually 160. In Sheffield, 184; but among the fork-grinders, the proportion is the appalling number 475; so that between these two periods,

three in this trade die to one in the kingdom generally.

Between the ages of 30 and 39, a still greater disparity presents itself. In the united kingdom, 136 only in the 1,000 die annually between these two periods. In Sheffield, 164; but in the fork-grinding branch, 410; so that between 20 and 40 years of age, in this trade, 885 perish out of the 1,000; while in the kingdom at large, only 296. Another step in the analysis, and we perceive that between 40 and 49, in the kingdom 126 die; in this town, 155; and in this branch, 115, which completes the 1,000. They are all killed off. For in carrying forward the inquiry we observe that between 50 and 59, in the kingdom, 127 die; and in Sheffield, 155; but among the fork-grinders, there is not a single individual left. After this period of life, there are remaining in the kingdom, of the 1,000, 441; and in the town, 339; but none in this branch of manufacture.

#### Radiographic Appearances:

In the first stage of silicosis the radiograph shows the presence of discrete nodular shadows, circular and at the most 2 millimetres in diameter. They may be partially distributed throughout the films, more widespread, or even generalized, but they remain discrete. Sometimes they are, in part, obscured by emphysema. In the second

stage the whole of both lung fields are occupied by nodular shadows, and there is some coalescence to form more or less dense opacities. In the third stage the radiographs indicate areas of massive consolidation. It is sometimes necessary to differentiate the X-Ray findings from those of chronic military tuberculosis, sarcoidosis, berylliosis, military carcinomatosis, siderosis, and the haemosiderosis of chronic heart failure.

In the present study, out of 96 cases 32 cases were having Pneumoconiosis. Total out of 32 cases of Pneumoconiosis Rosette formation seen in 28 persons (87.5%). Calcified shadow seen in 1 case (3.2%) micronodular shadow 1 case (3.2%) Hilar lymphadenopathy 1 case (3.2%).

#### Pulmonary Tuberculosis Supervenes:

Pulmonary tuberculosis may be present in any stage of silicosis. It may alter the symptoms, physical signs, radiological appearances and the whole course of the disease. It is the most frequent accompaniment of silicosis. Since tuberculosis of the lungs may simulate silicosis in radiographs, no diagnosis should ever be made exclusively on radiographic appearances.

#### Incidence of Silicosis:

In 1929 Sutherland and Bryson investigated the occurrence of silicosis in sandstone workers in England.



They examined clinically 454 workers, and of these 266 were selected for radiological examination. Judged by X-rays, 112 of these men had silicosis. The workers most affected are the stone masons and after them the quarrymen, rock-getters, planers and wallstone dressers. Men who dig tunnels, sewers and graves in sandstone may be affected too. The disease appeared to become more common after forty years of age, and after twenty years in the industry.

In 1929 Sutherland, Bryson and Keating investigated clinically 494 workers in granite, of whom 211 were examined radiologically. Judged by X-rays, thirty-six of these men had silicosis. Examination of the radiographs in the positive cases classed as silicosis shows that the prevalent type of the shadows indicating fibrotic changes is somewhat different from that found in workers exposed to silica dust in other industries. In the radiographs of granite workers there are usually the increased hilar, linear and reticular shadows, but instead of the discrete, dense nodules found to a greater or less extent over the whole of both lungs, there is a diffuse, cloudy or woolly effect with more or less definite fine or very fine nodules occurring in areas irregularly placed over the lungs. In the whole series the radiographs showing the most discrete nodules of the silicotic type is in the case of a man who worked for nine years as a driller on granite, but had been employed for eighteen years as a tin miner underground.

Amongst the other cases, who had been almost exclusively employed on granite, there appears to be some relationship between the composition of the rock and the character of the fibrosis, as shown on the radiograph.

In our study, out of 96 cases. Pulmonary tuberculosis seen in cases 41(42.7%), chronic bronchities with emphysematous changes 23(23.9%) cases, and Pneumoconiosis in 32(33.3%) cases. Rosette formation calcification were the common finding.

#### Different type of Fibrosis:

Clinical evidence of fibrosis, as distinguished from radiological evidence of silicosis, was found in 260 cases, or 52.6 per cent of the 494 workmen examined; but X-rays showed silicosis in 17 per cent only. Comparing the result of the medical examinations in the sandstone industry and the granite industry, the proportion of cases of fibrosis amongst sandstone workers was 59 per cent of those examined compared with 52.6 per cent in the case of granite workers. The proportion of cases of silicosis in sandstone workers was 42 per cent of those radiologically examined, and 17 per cent in the case of the granite workers. If fibrosis of the lungs, diagnosed by clinical examination, be regarded as representing a slighter or earlier involvement of the same character as silicosis, then it would seem that granites and the igneous rocks of



granite type produce less injury to the lungs than do the sandstone. Having regard to the appearances of the radiographs in the two series of workers, there is a probability of a difference in character of the types of fibrosis produced by the two kinds of dust, and this probability is increased by the proportion of cases of fibrosis in the granite series showing an approximation to that found in the sandstone series while the proportion of silicosis cases remains far behind (Middleton, 1930).

The clinical, radiological and pathological findings in workers exposed to dust of graphite have been described by Dunner (1945), Dassanayake (1948) and Harding and Oliver (1949). The radiological evidence of pulmonary changes often preceded subjective symptoms, and occurred after ten to fifteen years or more; it included scattered nodular shadows, and rounded conglomerate masses which varied in shape and size and, in some cases, were seen to contain cavities; their presence was confirmed by necropsy. The clinical picture was that of a slowly developing silicosis, whereas the radiological and pathological findings more closely resembled those of pneumoconiosis of coal miners; copious black graphite-containing liquid was found in the lungs, with numbers of graphite bodies resembling asbestosis bodies in general form. The large amount of carbon probably acted by upsetting the expulsive power of the lungs and thus allowed the quartz to exert prolonged localized action.

Macklin and Middleton(1923) carried out the first large-scale investigation into the chest condition of foundry workers in England. During a general enquiry directed especially into the health of metal grinders using sandstone wheels, they included in the survey examinations of 201 dressers of steel castings and found that 22.8 per cent had pulmonary fibrosis. Their work was done without the help of radiological and bacteriological examinations, their conclusions being made from clinical studies. In comparison, 73 per cent of a group of 495 wet sandstone grinders showed pulmonary fibrosis; the fettling of castings was therefore shown to be a less unhealthy job. It should be noted that at this time fettling was done mainly with hand tools; pneumatic tools came into more general use later. When the silicosis compensation schemes came to be formulated steel dressers, but not iron dressers, were scheduled as a group entitled to compensation for silicosis.

Harding, Gloyne and McLaughlin applied the term mixed dust pneumoconiosis or mixed dust fibrosis. The survey directed by Dr. A.I.G. McLaughlin was a concerted effort by employers, trade unions, Government departments, statisticians, pathologists, radiologists, clinicians and engineers. The investigations included the results of clinical and radiographic examinations of 3,059 workers in 19 foundries, an analysis of the records of lungs disease in foundry workers in the files of the Factory

Department and the Silicosis Medical Board, pathological investigations, of the lungs of 64 foundry workers and dust surveys in three foundries. As a result of this work, under the Iron and Steel Foundries Regulations, 1953, which became fully operative in 1956, iron foundry workers are protected by special regulations and paid benefit if they develop pneumoconiosis.

In present study the co-relation of Pneumoconiosis with durations of illness among 32 patients out of 96 cases studied give very interesting co-relation of duration of exposure. Those exposed for 5 years to 10 years showed incidence 25% in 8 cases only and those exposed for 10 years to 15 years having high incidence of 11 (34.4%) out of 32 cases and for those who exposed for 15 years to 20 years duration 13 (40.6%) highest. The clinical findings were also same in all the persons like chest pain, cough and expectoration and Dyspnoea were present cent percent. Haemoptysis were low only 10 (10.4%).

#### Complete occupational History:

In 1943 all the workers in the factory were interviewed and occupational histories taken. It was found that out of 163 men employed, twenty-two had worked in coal mines, and of these, nineteen showed radiographic evidence of either reticulation, nodulation or massive shadows, a

complicating factor not considered in certain reports on the disease. In 1944 further radiographs were made, and these revealed a man who had worked on the shredder for a year and who showed in his chest the radiographic changes.

Thus it is concluded by present study that patients who are working as stone cutters in stone grinding machines in stone crushers near by Jhansi area are definitely affected by the Pneumoconiosis. The long duration workers is more affected by Pneumoconiosis and complications of lungs.

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## S U M M A R Y

## S U M M A R Y   A N D   C O N C L U S I O N

The present study "A RADIOLOGICAL STUDY OF PNEUMOCONIOSIS IN PERSONS WORKING IN STONE CRUSHERS IN JHANSI DISTRICT" was carried out in the Department of Radiology, M.L.B. Medical College, Jhansi between July 1987 to August, 1988.

Patients selected were from working in stone crushers area near by Jhansi district. The person having duration of work in dust area 5 years or more were included in the study.

Total 96 persons working in stone crusher were studied, they were subdivided according to duration of exposures into 3 groups i.e.-

1. Duration of exposure 5 years to 10 years.
2. Duration of exposure 10 years to 15 years.
3. Duration of exposure 15 years to 20 years.

Detailed history includes symptomatology clinical examinations were taken. Each patient was investigated Radiologically for chest examination.

The standard technique was used for X-ray chest

PA view and lateral view. All the Radiological investigations were performed in the Department of Radiology, M.L.B. Medical College, Jhansi. The observations could be summarised as follow. The following conclusion could be made:

(1) The review the X-rays out of total 96 cases were having changes of Pneumoconiosis out of 96 cases studied. Pneumoconiosis was found in 32 cases (33.3%) chronic bronchitis was seen alongwith emphysema 23 cases (23.95%). Pulmonary tuberculosis was found to be in 41 cases (42.7%).

(2) Out of 32 cases of Pneumoconiosis, 8 cases were (25%) having duration exposure 5 - 10 years, 11 cases duration of exposure 10 - 15 years (34.4%) , 13 cases (40.6%) were having duration of exposure 15 - 20 years.

(3) Pneumoconiosis presented as Rosette formation in 28 cases (87.5%), calcification was seen only in 1 case (3.2%), micro-nodular shadows seen 1 case (3.2%). Lymph-adenopathy. (Hilar) seen in 1 case (3.2%). Non of the patient showed macro nodular shadows.

After reviewing the above finding following conclusion could be made.

(1) The duration of exposures very important for occurrence of Pneumoconiosis.

(2) Longer the duration the chances of Pneumoconiosis increase (round about two fold in 20 years as compare to 5 years).

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